Psoriatic Plaques "Koebnerizing" to Areas of Acanthosis Nigricans in an Obese Female

Clues to a Common Pathway?

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ABSTRACT

Recent evidence suggests that the activation of several growth factor receptors (EGFR, IGFR1, and FGFRs) is a possible cause of acanthosis nigricans, a skin disorder characterized by velvety thin plaques in skin folds and often seen in patients with insulin resistance. The authors report a 14-year-old obese (body mass index = 38.5kg/m^2) girl with a history of polycystic ovarian syndrome and pre-diabetes who presented with psoriatic plaques in her scalp and, subsequently, in areas mostly confined to where she had characteristic lesions of acanthosis nigricans. The authors propose that this as-of-yet unreported observation may represent a preferential koebnerization phenomenon where the abnormal keratinocyte proliferation in acanthosis nigricans may serve as the epidermal "micro-trauma" necessary to incite the prototypical isomorphic response seen in psoriasis. (*J Clin Aesthet Dermatol.* 2014;7(11):40–41.)

Acanthosis nigricans (AN) is a skin disorder characterized clinically by velvety, brown, thin plaques in skin folds, such as the axillae, antecubital fossae, or neck.[1] Histopathologically, the lesions demonstrate papillomatosis and hyperkeratosis.[2] These skin eruptions are usually associated with insulin resistance, obesity, and occasionally malignancies. The underlying mechanism for the skin findings is not completely understood; however, evidence suggests that the activation of several growth factor receptors (EGFR, IGFR1, and FGFRs) is the possible cause of AN.[3]

Psoriasis is a chronic T-cell mediated inflammatory skin condition prevalent in 0.1 to 2.8 percent of the population. The dermatological manifestation consists of salmon-pink plaques with overlying silvery-white scale. Among children, the extensors of the legs are the most common initial sites followed by the scalp.[4] Koebnerization is a classic sign and has been reported to occur in striae.[5] Recently, a focus has been placed on the exploration of an association between pediatric psoriasis severity and obesity.[6]

To the authors' knowledge, there have not been reports of psoriasis co-localizing preferentially to sites of acanthosis nigricans.

CASE REPORT

The authors report a 14-year-old obese (body mass index = 38.5kg/m^2) girl with a history of polycystic ovarian syndrome diagnosed by an endocrinologist, pre-diabetes (fasting glucose = 109), and acanthosis nigricans who presented for worsening psoriasis. The patient had a history of obesity since infancy and had developed psoriasis at the age of six. The psoriatic plaques had previously been mostly confined to the scalp for the last eight years and had not been previously treated. She denied rubbing or scratching the affected areas.

On physical exam, the patient had salmon-pink plaques with fine overlying silvery scales on her scalp that extended beyond the hairline. She also had a pink plaque in her intergluteal cleft circumscribed by hyperpigmented, velvety-to-verrucous plaques. Her neck, axillae, infra- and inter-mammary areas, umbilicus, abdominal folds, and the skin overlying the metacarpophalangeal joints had similar psoriatic plaques overlying lesions clinically consistent with acanthosis nigricans (Figures 1 and 2). The patient did not have geographic tongue, and her oropharynx was clear. She had no psoriatic nail changes or joint involvement. A thorough work-up for metabolic lab abnormalities was

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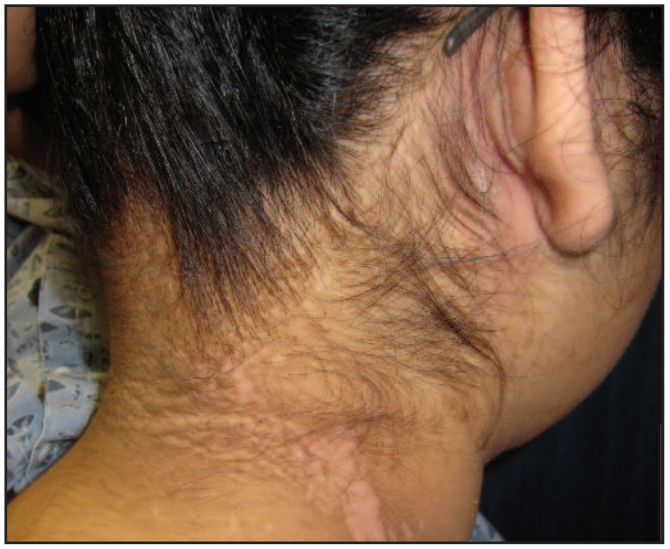

Figure 1. Psoriatic plaques overlying acanthosis nigricans of the neck

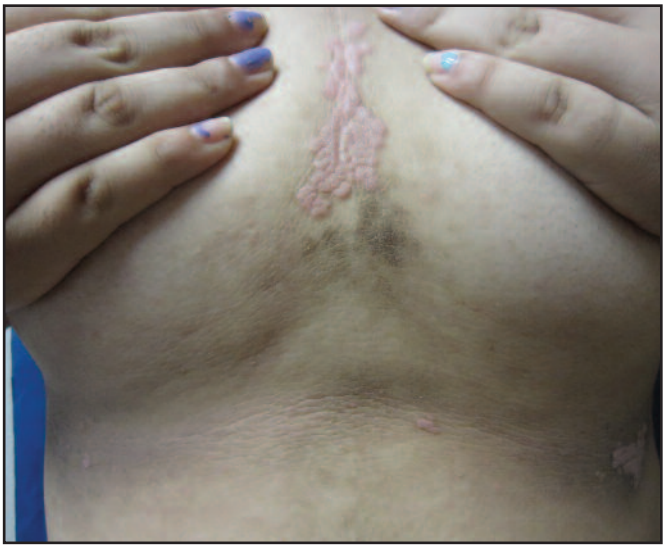

Figure 2. Psoriatic plaques and acanthosis nigricans along the inframmary fold and intermammary space

notable only for elevated insulin (51µU/mL; nl 2.6–25) and a mild transaminitis (AST 33 U/L; nl 15–30 and ALT 51 U/L; nl 5–30).

Her psoriasis was treated with topical fluocinonide 0.05% ointment for the body and ketoconazole 2% shampoo with fluocinonide 0.05% solution for the scalp. Dietary and lifestyle modifications were also recommended, and the patient was referred to pediatric endocrinology and pediatric liver team specialists.

DISCUSSION

The Koebner phenomenon, first described in 1872 as a hallmark of psoriasis, refers to an isomorphic response where there is a tendency for an established dermatosis to appear in areas of cutaneous trauma.[1] Excoriations or chronic rubbing could account for the observed koebnerization; however, the patient denied this behavior.

Alternatively, the authors suggest the possibility that at an ultra-structural level, the extensive papillomatosis and hyperkeratosis present in acanthosis nigricans could serve as a type of epidermal micro-trauma that under certain conditions may incite a prototypical isomorphic response. Studies have revealed increased IGF-1 and EGF receptor expression in psoriasis correlating with increased keratinocyte proliferation.[7] The authors propose that at the molecular level, overactive growth factor receptors in AN keratinocytes may set up a milieu of abnormal keratinocyte proliferation that could subsequently trigger eruptions of psoriatic plaques.

Although future studies are needed, this case of psoriatic plaques presenting preferentially in areas of AN in an obese adolescent potentially links AN and psoriasis both on a molecular biological and structural level. Thus, while Koebener co-localization of skin diseases has been well-described previously, the authors suggest that AN may be another condition that can co-localize with psoriasis.

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